ABSTRACT

Title of Dissertation: THE NEURAL CORRELATES OF SOCIAL MOTIVATION IN AUTISM SPECTRUM DISORDER DURING A REAL-TIME PEER INTERACTION

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Autism Spectrum Disorder (ASD) is characterized by difficulties with social motivation and social interaction. However, the neural underpinnings of these processes are poorly understood, and past studies investigating this subject have significant methodological limitations. This study is the first to investigate the neural correlates of social interaction in children and adolescents diagnosed with ASD using a naturalistic "chat" paradigm that mimics real-world reciprocal conversations. Despite core weaknesses in social interaction, participants with ASD showed similar brain activation to their neurotypical counterparts while initiating conversations and receiving replies from peers. Two notable group differences emerged, however. Participants with ASD showed blunted responses in the amygdala while initiating conversations and receiving replies, and they showed hyperactive responses in the temporal parietal junction (TPJ) while initiating conversations with peers. Findings

have implications for how we understand social motivational and social cognitive weaknesses in ASD.

THE NEURAL CORRELATES OF SOCIAL MOTIVATION IN AUTISM SPECTRUM DISORDER DURING A REAL-TIME PEER INTERACTION

by

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Dissertation submitted to the Faculty of the Graduate School of the
University of Maryland, College Park, in partial fulfillment
of the requirements for the degree of
Doctor of Philosophy
2018

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Acknowledgements

I wish to thank participants and their families for their significant time and effort in contributing to this research. I also would like to thank Dr. Redcay and current and former DSCN lab members—particularly Dustin Moraczewski, Katherine Warnell, Sydney Maniscalco, Eleanora Sadikova, Meredith Pecukonis, Zoey Maggid, Debbie Choi, Diana Alkire, and Aiste Cechaviciute—for assistance with study design, data collection, and data analysis. I am grateful to Wang Zhan and the Maryland Neuroimaging Center staff for their support during data collection. This study was supported by an NIMH R01MH107441 to E.R. Finally, many thanks to my family for all of their support during this long process.

Table of Contents

List of Tables

List of Figures

Introduction

Autism Spectrum Disorder (ASD) is characterized by difficulties with social interaction and social communication, including reduced interest in both approaching and sharing information with peers (American Psychiatric Association, 2013). A host of challenges are present throughout development in individuals with ASD that significantly impact social reciprocity. These include difficulties reading social and emotional cues (e.g., facial expressions, body posture), modulating behavior based on social context, and forming reciprocal relationships (Rutter & Schopler, 1987). Despite the centrality of social interactive difficulties in ASD, little is known about the neural correlates of social reciprocity in this clinical population. Investigating the neural correlates of social interaction in ASD will allow us to better understand the underlying reasons for atypicalities in ASD and may give insight into optimal interventions.

One factor that may contribute to these difficulties is reduced social motivation, although behavioral and neural evidence for this theory is mixed (Chevallier, Kohls, Troiani, Brodkin, & Schultz, 2012; Dichter & Adolphs, 2012; Jones, Gliga, Bedford, Charman, & Johnson, 2014, Kohls et al., 2012a). According to the social motivation theory, children with ASD view social stimuli as less rewarding than their neurotypical peers. As a result, they pay less attention to many aspects of the social world, including others' facial expressions and gestures (Chevallier et al., 2012; Dawson et al., 2002; Dawson, Bernier, & Ring, 2012; Dawson, Webb, & McPartland, 2005). Thus, children with ASD miss out on important social cues early in life, which may begin a developmental cascade that leads to greater social

disability and may change the course of brain development (Klin, Jones, Schultz, & Volkmar, 2003; Pelphrey, Shultz, Hudac, & Vander Wyk, 2011; but see Jones et al., 2014). In fact, increasing the number of self-initiated social encounters (i.e., motivation to interact with others) is considered a pivotal area of intervention in ASD, supporting the centrality of social motivational difficulties in the disorder (Koegel, Koegel, & McNerney, 2001; Koegel & Mentis, 1985).

Past research suggests that social cognitive, in addition to social motivational, weaknesses may underlie difficulties with social interaction in individuals with ASD (Baron-Cohen, 2000; Baron-Cohen, Leslie, & Frith, 1985; Travis & Sigman, 1998). While some individuals with ASD are unmotivated to seek friends, many report wanting friendships and romantic relationships (Henault & Attwood, 2002) but are unsuccessful at initiating and maintaining reciprocal relationships. This points to the possibility that for some individuals with ASD, social cognitive rather than social motivational weaknesses may be primary (i.e., they may *want* relationships but do not have the social skills to acquire them). It is possible that, as the result of many failed social interactions due to social cognitive weaknesses, people with ASD are then less motivated to further pursue social relationships. These social cognitive and social motivational challenges contribute to many autistic individuals reporting social anhedonia (i.e., the inability to derive enjoyment from interacting with others; Berthoz, Lalanne, Crane, & Hill, 2013; Carre et al., 2015; Chevallier et al., 2012), and half of adults with ASD reporting that they have no friends (Howlin, Goode, Hutton, & Rutter, 2004). In sum, throughout development, individuals with ASD demonstrate

social cognitive weaknesses coupled with diminished social motivation, which results in difficulties with social reciprocity and social interaction.

Brain regions involved in social motivation have been well characterized in typical development and include subcortical regions such as the amygdala and ventral striatum (VS), as well as orbitofrontal cortex (OFC), ventromedial prefrontal cortex (vmPFC), medial prefrontal cortex (mPFC), and anterior cingulate cortex (ACC) (Chevallier et al., 2012; see Berridge & Kringelbach, 2008; Haber & Knutson, 2010; Fareri, Martin, & Delgado, 2008; Taber, Black, Porrino, & Hurley, 2012 for reviews of reward processing). Consistent with behavioral evidence, the few studies that have investigated the neural correlates of social motivation in ASD show many inconsistencies.

There is some evidence that the neural systems involved in social motivation differ in autistic individuals compared to their neurotypical peers (Kohls et al., 2012; Richey et al., 2012; Scott-Van Zeeland, Dapretto, Ghahremani, Poldrack, & Bookheimer, 2010). While some studies find hypoactivation in ASD in VS (Richey et al., 2012; Scott-Van Zeeland et al., 2010), other studies evidence typical levels of VS activation (Delmonte et al., 2012; Dichter, Richey, Rittenberg, Sabatino, & Bodfish, 2012). While one study found hypoactivation in ASD in amygdala and ACC to social rewards (Kohls et al., 2012b), other studies found no differences between groups in these regions (Delmonte et al., 2012, Dichter et al., 2012; Richey et al., 2012; Scott-Van Zeeland et al., 2012). Another study found no differences between NT and ASD in any of these classic reward regions in response to social rewards (Delmonte et al.,

2012), and one study found *hyper*activation in ASD in amygdala (Dichter et al., 2012). There are several possible explanations for these inconsistencies.

Critically, social motivation in autism has not been studied in a social interactive context, which is problematic for two reasons. First, the lack of reciprocal social interaction and real-world applicability in past studies is troubling since individuals with ASD have the most difficulty in interactive social communicative contexts but may perform within normal limits on non-interactive laboratory tasks related to social cognition (Chevallier et al., 2015; Schilbach et al., 2013; Senju, Southgate, White, & Frith, 2009). Second, failing to study social motivation in a social interactive context might underestimate the role of social cognitive neural circuitry. In fact, recent studies have found that embedding participants in social interactive contexts recruits brain regions involved in social cognition, even in the absence of a social task (Rice & Redcay, 2016). Thus, to capture meaningful differences between groups and to better understand the neural correlates of social motivation and social cognition, it is imperative to use ecologically valid, social interactive paradigms.

A recent study with a more engaging, interactive social paradigm (i.e., playing a domino game with a "live" partner) investigated the neural correlates of social motivation in ASD (Assaf et al., 2013) and found involvement of both social motivational and social cognitive brain regions. This study found hypoactivation in VS in the ASD group, consistent with some past research (Richey et al., 2012; Scott-Van Zeeland et al., 2010). By embedding the rewards in a social context, this study also found activation in both the ASD and NT groups in the following social

cognitive regions when participants believed they were playing the game with another person: bilateral temporal parietal junction (TPJ), superior temporal sulcus (STS), middle temporal gyrus (MTG), and medial prefrontal cortex (mPFC). When the ASD and NT groups were compared directly, there was MTG hypoactivation in ASD. While Assaf et al. (2013) improved upon past social reward paradigms by embedding rewards in a social context, the reward itself (scoring points in the game) was nonsocial. Thus, the VS hypoactivation in ASD was not related to social interaction per se. Additionally, participants were unable to communicate directly with the other person and could not share information with them or learn facts about them. Instead, participants were competing with the other person in a game. While the context was social (because participants believed they were playing with a real person), it was not particularly interactive and it was dissimilar from everyday reciprocal conversations with peers. For example, in everyday reciprocal conversations, we share and receive information from our conversational partners—we are truly *interacting* with them.

To overcome these limitations, our lab developed a study that embedded typically developing children in a real-world social communicative interaction, where both initiating and responding to bids for social interaction could be investigated (Warnell, Sadikova, & Redcay, 2017). Participants believed they would be chatting with a same-aged peer and sharing information about themselves as well as learning information about their peer. Briefly, findings revealed classic reward regions during social initiation and both reward and social cognitive regions during peer reply. This finding suggests that embedding children in a naturalistic, reciprocal social interaction recruits social cognitive brain regions, unlike past social motivation

studies that did not involve social interaction. Thus, this study design was critical to understand the neural correlates of social interaction and allowed us to tap into both social motivational and social cognitive neural circuitry.

The purpose of the current study was to capitalize on the experimental strengths of Warnell et al. (2017) to investigate the neural correlates of social interaction in children with ASD between the ages of 7 and 14. Because a reciprocal social interaction involves two stages—sharing information with peers (e.g., self-disclosure, initiation of a topic) and receiving a response from the peer—we sought to understand what brain regions were involved in each of these stages. This is important given that the neural substrates of initiating and receiving a response may be different both in NT and ASD (Haber & Knutson, 2010; Kohls et al., 2012a). Finally, given that it may be rewarding for children to receive any type of contingent response, we investigated what brain regions were active when children received a social contingent response (i.e., from a peer) versus a non-social contingent response (i.e., from a computer).

The current study had three aims. 1) To determine differences between ASD and NT in the neural correlates of initiating a social interaction with a peer (i.e., sharing information about themselves) versus sharing information about themselves with a computer 2) To determine differences between ASD and NT in the neural correlates of receiving a response from a peer (i.e., learning something about their conversational partner) versus receiving a response from the computer; and 3) To determine differences between ASD and NT in the neural correlates of social engagement during an interaction (i.e., [learning something about their conversational

partner versus receiving a response that the partner is away] versus [receiving a response from the computer versus the computer showing a 'disconnected' message]).

We hypothesized that, compared to NT children, children with ASD would show fewer differences in VS between sharing information about themselves with a peer and a computer. We expected that in ASD, the VS would be active to both conditions but would not activate more to the peer vs. the computer condition. This would suggest that the neural circuitry supporting motivation in ASD is intact, but that sharing information in a nonsocial condition is as rewarding as sharing with a peer.

We also predicted that, compared to NT children, children with ASD would show dampened neural activation to peer responses in both reward (VS, OFC, dmPFC) and social cognitive (TPJ, STS) regions. We predicted that while the ASD within-group analysis would show higher activation in reward and social cognitive regions to peer responses, the between-group analysis contrasting ASD with NT would reveal less activation in the ASD group to the main effect of partner type (Peer vs. Computer). This would suggest that the neural circuitry supporting the second stage of social interaction—receiving a response from a peer—is underactive in ASD.

Finally, we hypothesized that, compared to NT children, children with ASD would show less activation in social cognitive and reward regions for social engagement trials, i.e., when the peer responds versus is away or when the peer responds versus when the computer responds. We predicted that within the ASD group, there would be differences between peer response vs. away in social cognitive

and reward regions, but that the between-group analysis would reveal less activation in the ASD group. This would suggest that the neural circuitry supporting social engagement is underactive in ASD.

Methods

Participants

Participants included children and adolescents between the ages of 7 and 14 diagnosed with an ASD (confirmed in our lab with an Autism Diagnostic Observation Schedule, Second Edition; ADOS-2 and the Social Communication Questionnaire; SCQ). All recruited participants had a clinical diagnosis of ASD prior to participating in our study, and all participants met criteria for ASD during our ADOS-2 assessment, which was conducted by a research-reliable administrator. Participants were recruited from a database of local families and from flyers posted on campus and at local businesses and doctors' offices serving individuals with ASD. Participants were eligible for the study if they were not more than 5 weeks premature (>35 weeks gestation), were native English speakers, and had no history of head injury, seizures, or any other characteristics that would prevent MRI scanning. We recruited 28 participants, and due to excessive scan motion from 8 participants, our final sample included 20 participants (2 females). A mean age-, mean IQ-, and sex-matched group of NT participants was used as the comparison group for analyses comparing ASD and NT. NT participants had no family history of ASD.

Task procedures: setting up the chat

Peer trials. Prior to entering the MRI scanner, the experimenter explained to participants that they would be chatting with another child in a separate research lab somewhere in the United States. In reality, the experimenters had preprogrammed all "peer" responses to maintain experimental control. Participants learned that they

would use “yes” and “no” buttons to share information about themselves (e.g., hobbies, interests) with the other child, and would also learn information about the other child. To begin interacting with the other child, participants learned that they would answer “yes” or “no” to a statement such as, “I play soccer” (Peer Initiation). The other child would see the participant’s answer and would respond by saying, “Me too/neither!” or “That’s not what I picked” (Peer Engagement). Importantly, the peer primarily responded with the same answer as the participant (“Me too/neither”), but participants also viewed two Disagreement trials (i.e., “That’s not what I picked”) to increase believability of the peer’s responses. These Disagreement trials were not analyzed. Participants learned that the other child would not always be available to chat with the participant, because they sometimes had to play another game. This was a cover story in order to have non-contingent social trials (i.e., trials for which the peer did not reply to the participant). For those trials, an away message was displayed as the peer response (i.e., “I’m away”); however, the other child could still see the participant’s answers to the self-relevant statements. This was the Peer Non-Engagement condition. In sum, each time the participant learned that they would share an answer with the other child, initiating a conversation with the other child that could either be responded to or not responded to, depending on if the other child was available or busy playing his or her other game.

After the experimenter confirmed that the participant understood the instructions, he or she took a photograph of the participant and pretended to email it to the lab where the other child was located. Participants were informed that soon they would be able to see photographs of potential chat partners. Next, participants

viewed a preprogrammed series of screens that matched them with the other child and created the chat illusion. Participants viewed two photos of age- and sex-matched peers (smiling, direct gaze photos from the NIMH Child Emotional Faces Pictures Set; Egger et al., 2011) that the computer had matched them with, and to increase motivation, participants had the opportunity to choose the peer with whom they would like to chat (c.f. Guyer et al., 2009). Participants then rated this peer on a 1-5 scale in terms of how interested they were in chatting with the peer and how much they thought they would like the peer in real life.

Computer trials. Participants learned that sometimes, they would just be connected to a computer (and not another child). During these trials, no one would see the participant's answers to the self-relevant statements (Computer Initiation), and the computer would generate a response ("yes" or "no") randomly, by spinning a wheel. The participant then would see the computer's response ("Matched" or "Mismatched"), with "Matched" meaning that the computer randomly generated the same response as the participant, and "Mismatched" meaning that the computer randomly generated the opposite response. This was the Computer Engagement condition. Importantly, the computer would primarily respond with "Matched," but participants would also view two Disagreement trials (i.e., "Mismatched") to increase believability of the computer's responses. These trials were not analyzed. Participants learned that at times, the computer would lose its connection, and when this happened, the participant's response would not be sent to the computer, and the computer would send the message "Disconnected." This was a cover story in order to have non-contingent nonsocial trials (Computer Non-Engagement condition). In

sum, each time the participant shared an answer with the computer, the participant's answer was either responded to or not responded to, depending on if the computer was connected or disconnected. See Figure 1 for a diagram of the task.

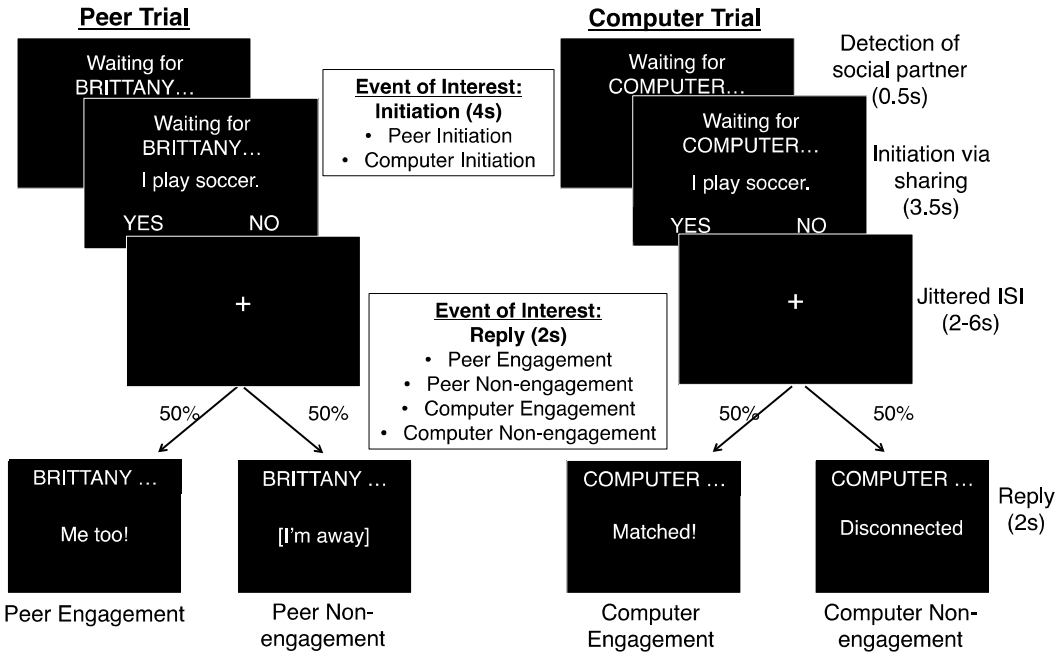


*Figure adapted with permission from Warnell et al. (2017)

Stimuli characteristics

The chat partner photographs of age- and gender-matched peers have been used in past studies to assess perception of peers in middle childhood (Guyer et al., 2009, 2012, 2014). The self-relevant statements (e.g., "I like French fries") to which participants responded were developed in our lab by piloting a sample of 12 NT

children between ages 7 and 12. We piloted 168 statements and excluded items that had the lowest response rates within the response window. We then selected eight statements to which children answered "Yes" and "No" approximately 50% of the time each. We chose these statements as Disagreement items, since it would be plausible for a peer to disagree with the participant's response (e.g., "That's not what I picked"). Disagreement items were included to increase believability of the chat; however, these trials were not included in the analysis of fMRI data due to their low frequency. The final 96 selected statements were matched between conditions on average reaction time, response rate, and answer ("yes" vs. "no").

Stimuli presentation

The task was programmed and presented using the Psychophysics Toolbox Extension for MATLAB 7.6 (PTB-3; Brainard, 1997). Participants viewed 52 trials of each initiation type (Peer Initiation, Computer Initiation) and 24 individual trials of each analyzed reply type (Peer Engagement, Peer Non-engagement, Computer Engagement, Computer Non-engagement). If participants did not respond "yes" or "no" to the self-relevant statement (e.g., "I play soccer") within the response window, they saw a non-engaged reply and these trials were not analyzed. We determined the distribution of the trial types and the timing of the jitters and inter-trial intervals using the program OptSeq (http://surfer.nmr.mgh.harvard.edu/optseq/) to ensure the optimal timing in order to allow for independent analysis of the events versus baseline. Specifically, Initiation and Reply types were counted together as one trial for event spacing and ordering (e.g., Peer Engagement was one trial type). This model was

further tested for collinearity using AFNI's 3dDeconvolve (Cox, 1996; http://afni.nimh.nih.gov/), ensuring that all beta values of interest were estimable.

We presented a 2-6s jittered fixation cross, distributed exponentially and centered around 3.5s, between the Initiation and Reply portions and between each trial and for the first 15s and last 10s of each run. In between each run, children were shown the photo of the age- and sex-matched peer that they had selected in the portion of the experiment before entering the scanner (Egger et al., 2011), along with a message about the chat (e.g., "That was fun!"). The purpose of this was to reinforce the live chat illusion.

We assigned each participant to one of four stimuli sets, which differed based on the assignment of the 96 items to the different types of trials (Peer Engagement, Peer Non-Engagement, Computer Engagement, and Computer Non-Engagement). The order and timing of the trial types within each run were determined prior to the scan. Trials were randomly assigned to different positions within the runs (e.g., the Peer Engagement item selected could be any of the 24 possibilities), and the participant's stimuli set (i.e., which items were assigned to which condition) and run order were predetermined to ensure that all possibilities were represented. This was an important step to ensure that reaction time and response rates, as determined from the pilot data used to select stimuli, were matched for the peer versus computer conditions for all participants.

Image acquisition and preprocessing

Functional magnetic resonance imaging data was collected using a 32-channel head coil on a single Siemens 3.0-T scanner at the Maryland Neuroimaging Center

(MAGNETOM Trio Tim System, Siemens Medical Solutions). The scanning protocol for each participant consisted of four runs of the experiment (T2*-weighted echo-planer gradient-echo; 40 interleaved axial slices; voxel size=3.0 x 3.0 x 3.0 mm; repetition time=2200ms; echo time=24ms; flip angle=78°; pixel matrix=64 x 64), and a single structural scan (three-dimensional T1 magnetization-prepared rapid gradient-echo sequence; 176 contiguous sagittal slices, voxel size=1.0 x 1.0 x 1.0 mm; repetition time=1900ms; echo time=2.52ms; flip angle=9°; pixel matrix= 256 x 256). Preprocessing of fMRI data was performed using AFNI (Cox, 1996). Data was slice time corrected and then aligned to the first volume (using a rigid-body transform). The anatomical scan was aligned to the first volume and then transformed to MNI space via linear and non-linear transformations. We then normalized the functional data using these same transformation parameters, and spatially smoothed with a 5mm full-width half-maximum (fwhm) Gaussian kernel. Functional data was intensity normalized so that each voxel would have a mean of 100. Volumes in which the difference between two consecutive volumes exceeded 1mm (across translational and rotational movements) were considered outliers and were censored in subsequent analyses. Runs were excluded if total frame deviation was greater than 4mm in any direction, or if greater than 10% of collected volumes were identified as outliers. Participants with at least three usable runs were included in the final analyses. Five participants in the ASD sample had motion spikes greater than 4mm at the very end of a run. In these instances, we were able to exclude a minimal number of data points while still keeping the majority of the run in our analyses. Importantly, the number of useable runs did not differ between NT and ASD groups ($t(38)=0$, $p=1.0$). The mean

number of runs for NT participants and ASD participants was identical: 3.65 runs. Additionally, the average mean frame displacement ($t(38)=1.52$, $p = 0.14$) and average max frame displacement ($t(38)=1.26$, $p = 0.21$) across useable runs was not significantly different between groups.

Post-scan questionnaire

Immediately following the scan, an experimenter verbally administered a questionnaire to participants. The purpose of this questionnaire was to assess participants' subjective experiences of the chat. A subset of questions assessed levels of enjoyment during the two Initiation types (Peer vs. Computer) and the four Reply types (Peer Engagement, Peer Non-engagement, Computer Engagement, Computer Non-engagement). Additionally, the experimenter asked participants how much attention they paid when initiating, as well as how much they wanted to see the answers of the peer versus the computer. Questions were presented on a 1 to 5 point Likert-type scale. Finally, we assessed participants' belief in the live illusion (See Appendices for full post-scan questionnaire).

Data Analysis

After preprocessing fMRI data using the procedures discussed above, we ran Ordinary Least Squares regression analyses for each participant's concatenated runs with regressors for each condition (Peer Initiation, Computer Initiation, Peer Engagement, Peer Non-engagement, Computer Engagement, Computer Non-engagement) as well as nuisance regressors. Nuisance regressors included baseline and linear, quadratic, and cubic trends in addition to twelve motion regressors (i.e.,

the frame deviation at each volume for the six directions of translational and rotational motion and their derivatives). Regressors for all conditions were created by convolving a gamma-variate basis function with the stimulus timing function (duration the length of one event and an amplitude of 1). Contrasts were estimated for each condition of interest and for the following comparisons: 1. Initiation: Peer vs. Computer; 2. Reply: Main effect of Peer versus Computer [(Peer Engagement + Peer Non-engagement) > (Computer Engagement + Computer Non-Engagement)]; 3. Reply: Effect of social engagement [(Peer Engagement versus Peer Non-Engagement) versus (Computer Engagement versus Computer Non-Engagement)]; and 4. Other Reply contrasts of interest: Peer Engagement versus Peer Non-Engagement and Peer Engagement versus Computer Engagement.

Whole-brain analysis. We then incorporated coefficients and t-statistics for each contrast in the group-level analyses using mixed effect models (3dMEMA; Chen, Saad, Britton, Pine, & Cox, 2013), modeling within- and between-subject variance. For each contrast, we calculated within- and between-groups (i.e., ASD versus NT) effects across all participants for each voxel using mixed effect models. Across comparisons, all contrast maps were thresholded at $p < .005$ and cluster corrected at $p < .05$ ($k=73$, using the updated version of AFNI's 3dClusStim; Eklund, Nichols, & Knutsson, 2015).

Region of interest (ROI) analysis. In addition to whole-brain comparisons, we conducted region of interest (ROI) analyses. Given our a priori hypotheses about the role of VS and amygdala in social motivation and engagement, we selected two VS ROIs, bilateral inferior VS (corresponding to nucleus accumbens) and bilateral

superior VS (corresponding to ventral caudate), and one bilateral amygdala ROI for analysis. Ventral striatum ROIs were anatomically defined (Di Martino et al., 2008) and have been used in other studies investigating VS activation (e.g., Kelly et al., 2009; Kolla et al., 2016; Padmanabhan, Lynn, Foran, Luna, & O'Hearn, 2013). We chose to focus on ventral (as opposed to dorsal) striatum given that past studies have identified VS as important in processing social rewards (Izuma, Saito, & Sadato, 2010; Pfeiffer et al., 2014; Schilbach et al., 2010; Tamir & Mitchell, 2012). The amygdala ROI was anatomically defined with the Harvard-Oxford subcortical atlas (https://fsl.fmrib.ox.ac.uk/fsl/fslwiki/Atlases).

In addition to the VS and amygdala ROIs, we also selected four social cognitive ROIs: bilateral anterior STS, bilateral TPJ, mPFC, and dmPFC. We selected these regions due to their involvement in previous studies investigating social interaction and social cognition (Assaf et al., 2013; Warnell et al., 2017; Saxe, 2009). Given that these brain regions are difficult to anatomically define, we functionally defined them based on a meta-analysis of functional brain imaging studies related to theory of mind (Schurz, Radua, Aichorn, Richlan, & Perner, 2014). While this meta-analysis was conducted in adults as opposed to children, it is the largest meta-analysis of its kind, summarizing 73 studies and 1241 participants. Thus, activation foci are more reliable than had we used a single study with a child or adolescent sample. Moreover, the activation foci selected from Schurz et al. (2014) are similar to those in a theory of mind study that was conducted in a similarly-aged child sample as the current study (Gweon, Dodell-Feder, Bedny, & Saxe, 2012). For each ROI, we drew a sphere with a 6mm radius around the peak voxel identified in Schurz et al. (2014)

then extracted individual participant beta values from these functionally defined regions. For all ROIs, we analyzed effects during the Initiation period using 2 (Peer vs. Computer) × 2 (NT vs. ASD) ANOVAs, and we analyzed effects during the Reply period using 2 (Engagement vs. Non-engagement) x 2 (Peer vs. Computer) x 2 (TD vs. ASD) repeated measures ANOVAs.

Results

Descriptive Analyses

NT and ASD participants did not significantly differ on age ($t(38) = 0.93$, $p = 0.36$), Verbal IQ ($t(38) = -1.78$, $p = 0.08$), Nonverbal IQ ($t(38) = -0.32$, $p = 0.75$), or Full Scale IQ ($t(38) = -1.51$, $p = 0.14$). For the participants with ASD, ADOS-2 scores were, on average, in the "Moderate" range. See Table 1 for descriptive statistics.

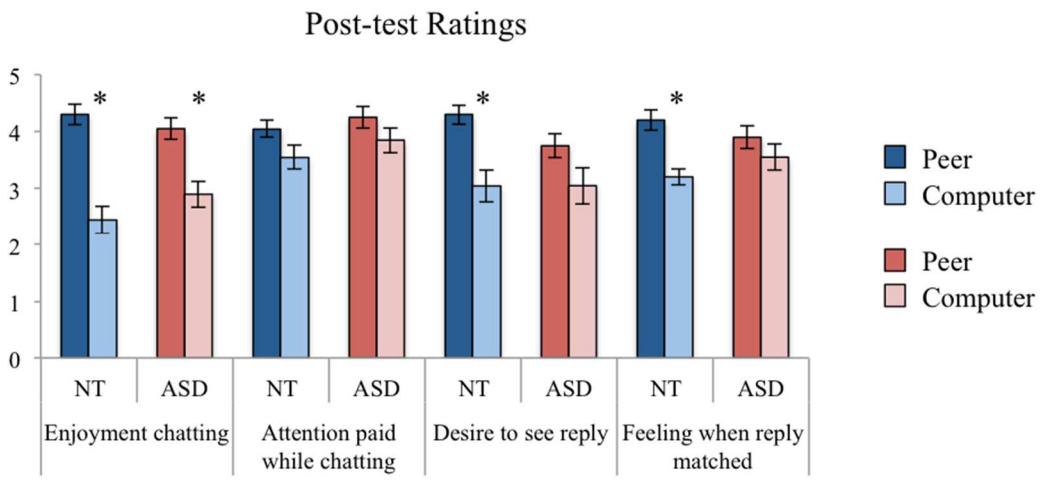
Table 1. Descriptive Statistics

	ASD	NT
N	20	20
Age	12.22 (1.81)	11.69 (1.73)
Sex	2 females	2 females
Nonverbal IQ	112.95	114.35
Verbal IQ	105.45	116.05
Full-Scale IQ	110.75	117.80
ADOS-2 Total	6.50 (1.67)	N/A
ADOS-2 Social	6.75 (1.52)	N/A
ADOS-2 RRB	6.55 (1.99)	N/A

Behavioral Analyses

Post-scan questionnaire. All children believed they were chatting with a real peer and both NT and ASD participants reported that they found the peer likeable (NT average rating: 4.1/5; ASD average rating: 3.95/5). Across the four questionnaire categories (i.e., enjoyment chatting, attention paid when chatting, desire to see reply, and feeling when the reply matched) a 2 (Peer vs. Computer) × 2 (NT vs. ASD) ANOVA revealed a significant main effect of partner type ($F(1,136) = 85.19$, $p < 0.001$), with higher ratings for the Peer. There was also a group by partner type interaction ($F(1,136) = 6.57$, $p = 0.01$). Post-hoc Tukey's Honest Significant Difference (HSD) tests revealed that both NT ($p < 0.001$) and ASD ($p = 0.001$) participants enjoyed chatting with the Peer more than the Computer, both groups of participants reported having equal attention while chatting with the Peer and Computer, only NT participants reported a stronger desire to see Peer (versus Computer) replies ($p = 0.005$), and only NT participants reported feeling better when their response matched the Peer's (versus Computer's) reply ($p = 0.002$) (Figure 2).

Figure 2. Both NT participants and those with ASD rated the Peer higher than the Computer in the following domain: enjoyment chatting. Only NT participants rated the Peer higher than the Computer in the following domains: desire to see reply and feeling when the reply matched.



fMRI analyses

Behavioral Data from Scan. A 2 (Peer vs. Computer) × 2 (NT vs. ASD) ANOVA revealed no significant main effects or interactions. While in the scanner, NT and ASD groups did not significantly differ on reaction time for answering Peer versus Computer questions ($t(28) = -0.13$, $p = 0.90$), percentage of skipped questions for Peer versus Computer ($t(32) = -0.15$, $p = 0.88$), or the proportion of time they answered "yes" to Peer versus Computer questions ($t(33) = 0.33$, $p = 0.74$).

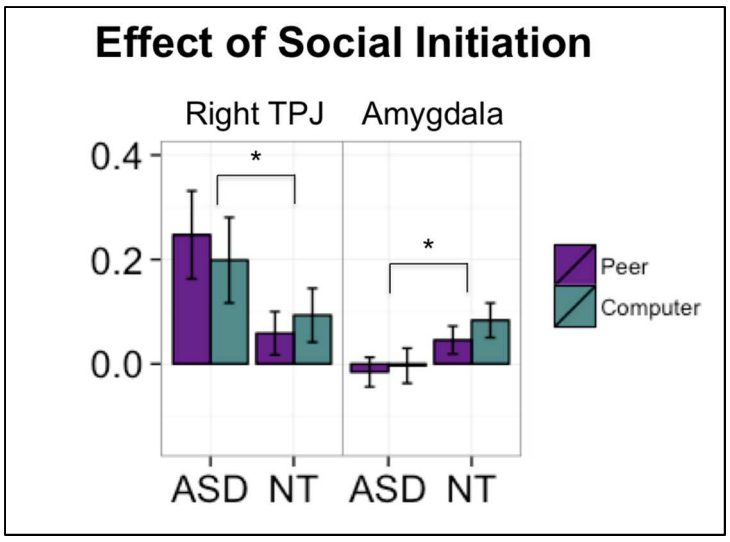
Within groups, participants answered Peer questions equally quickly to Computer questions (NT: $t(19) = -1.35$, $p = 0.19$, ASD: $t(19) = -0.84$, $p = 0.41$), they skipped the same proportion of Peer questions and Computer questions (NT: $t(19) = 0.05$, $p = 0.96$, ASD: $t(19) = -0.15$, $p = 0.89$), and they answered "yes" the same percentage of time to Peer and Computer questions (NT: $t(19) = 0.18$, $p = 0.86$, ASD: $t(19) = 0.52$, $p = 0.61$).

Effect of Social Initiation. Whole-brain analyses revealed no significant differences between ASD and NT groups when sharing information (i.e., initiation) with a peer versus sharing information with a computer. Consistent with Warnell et al. (2017), which included a subset of the NT participants included in the current study, there were also no differences in whole-brain activation between Peer versus Computer initiation within either group.

In addition to whole-brain analyses, we conducted region of interest (ROI) analyses by extracting participants' beta values from three anatomically defined ROIs

(amygdala, VSi, and VSs) and four functionally defined ROIs (TPJ, aSTS, mPFC, and dmPFC). Within the amygdala, a 2 (Peer vs. Computer) × 2 (NT vs. ASD) ANOVA revealed a significant main effect of group ($F(1,76) = 5.89$, $p = 0.02$). Specifically, NT participants showed greater activation across both conditions (Peer Initiation and Computer Initiation) compared to participants with ASD ($t(77) = -2.45$, $p = 0.02$). Within the right TPJ, a 2 (Peer vs. Computer) × 2 (NT vs. ASD) ANOVA also revealed a significant main effect of group ($F(1,76) = 4.77$, $p = 0.03$), but with ASD participants showing greater activation across both conditions compared to their NT peers ($t(61) = 2.21$, $p = 0.03$). There were no significant main effects or interactions within the other ROIs. See Figure 3 for graphs of the amygdala and right TPJ ROIs.

Figure 3. Effect of social initiation: ROI analysis. In the right TPJ, participants with ASD showed greater activation across both conditions compared to NT participants. In the amygdala, NT participants showed greater activation across both conditions compared to participants with ASD.



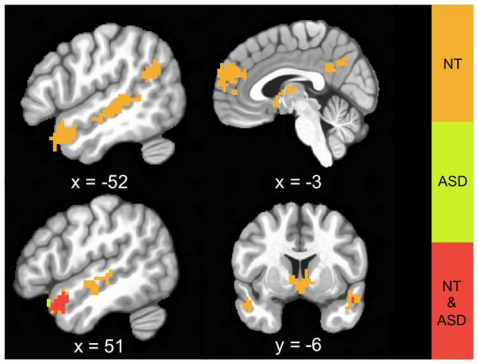
Effect of Mutual Engagement: Whole Brain Analyses.

Main effect of Peer versus Computer. Whole-brain analyses revealed no significant differences between ASD and NT groups when receiving a reply from a peer versus receiving a reply from a computer [(Peer Engagement + Peer Non-Engagement) > (Computer Engagement + Computer Non-Engagement)]. Each group, however, did show neural sensitivity to Peer versus Computer replies. Within the NT group, receiving a reply from a peer resulted in significantly higher activation in the following brain regions associated with social cognitive and reward processing: bilateral aSTS, bilateral STS, mPFC, posterior cingulate, TPJ, and caudate. Within the ASD group, receiving a reply from a peer resulted in significantly higher activation in left aSTS. See Table 2 for a list of brain regions showing greater activation for Peer > Computer replies. Figure 4 depicts distinct and overlapping activation for the two participant groups.

Table 2. Whole-brain main effect of Peer > Computer replies: peak t values, coordinates, and number of voxels ($p < 0.005$, $\alpha = 0.05$, k = 73 voxels).

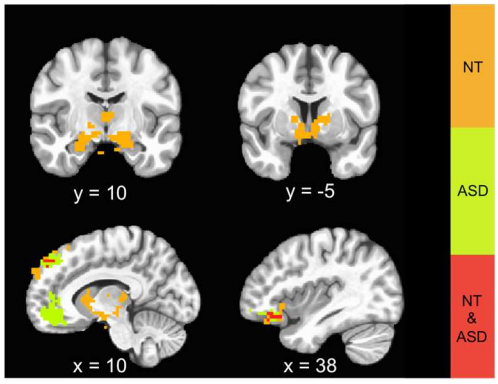
Region	Peak t	Peak x	Peak y	Peak z	# Voxels
NT					
Left aSTS	5.81	-60	-18	-18	199
Right aSTS	3.21	48	-24	-27	114
Left STS	3.67	-69	27	-12	143
Right STS	3.81	63	0	-12	115
mPFC	3.25	-3	-66	27	279
Posterior cingulate	3.91	-3	63	36	126
Left TPJ	3.10	-60	60	18	73
Caudate	3.16	0	-6	-12	202
ASD					
Right aSTS	4.18	51	-21	-24	74

Figure 4. While there were no significant differences between groups for the main effect of partner type at the whole-brain level, NT participants (orange) showed activation in a larger number of regions than participants with ASD (green) when chatting with the peer. The two participant groups had overlapping activation (red) in right aSTS.



Peer Engagement versus Peer Non-Engagement. There were also no significant group differences when participants received a "Me too!" reply from the peer versus when they received an "I'm away" reply from the peer. However, each group did show neural sensitivity in reward regions to this contrast. Within the NT group, there was greater activation in response to peer engagement in caudate, bilateral amygdala, right IFG, and dmPFC. Within the ASD group, there was greater activation to peer engagement in vmPFC, right IFG, and dmPFC. Figure 5 depicts distinct and overlapping activation for the two participant groups.

Figure 5. While there were no significant differences between groups for the contrast of Peer Engagement > Peer Non-Engagement at the whole-brain level, NT participants (orange) showed activation in a larger number of regions than participants with ASD (green). The two participant groups had overlapping activation (red) in dmPFC and right IFG.



Peer Engagement versus Computer Engagement. The comparison of receiving a "Me too!" response from a peer versus receiving a "Matched!" response from the computer also revealed no group differences. However, NT participants showed greater activation to the peer in mPFC, posterior cingulate, and caudate, while no areas of the brain were more active to peer versus computer in ASD participants. See Table 3 for a list of brain regions showing greater activation for Peer Engagement > Computer Engagement.

Table 3. Whole-brain contrast of Peer Engagement > Computer Engagement: peak t values, coordinates, and number of voxels ($p < 0.005$, $\alpha = 0.05$, k = 73 voxels).

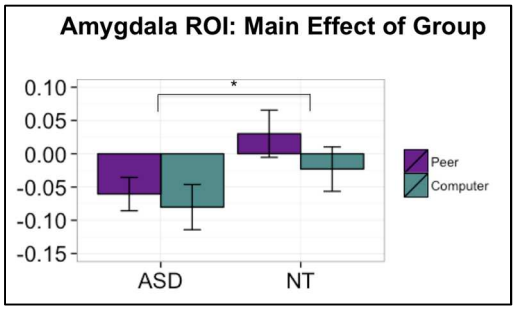
Region	Peak t	Peak x	Peak y	Peak z	# Voxels
NT					
mPFC	4.29	0	-63	24	256
Posterior cingulate	3.96	-3	60	36	150
Caudate	3.20	0	-6	-15	307
ASD					
NONE					

Effect of Social Engagement. Finally, we were interested in the effect of social engagement, or receiving a "Me too" reply from the peer versus an "I'm away" reply from the peer VERSUS receiving a "Matched!" reply from the computer versus a "Disconnected" reply from the computer. At the whole brain level, there were no significant group differences. Within each group separately, there were also no brain regions that showed significant activation for this contrast.

Effect of Mutual Engagement: ROI Analyses. For ROI analyses, we conducted a 2 (Engagement vs. Non-engagement) x 2 (Peer vs. Computer) x 2 (TD vs. ASD) repeated measures ANOVA for each ROI. Several regions showed a main effect of

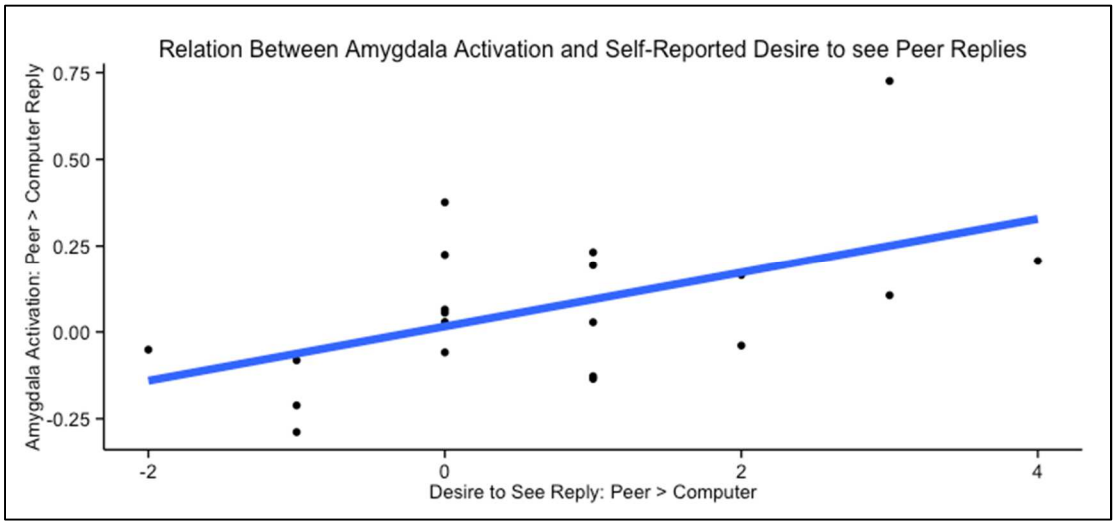
Engagement. These included amygdala ($F(1,89) = 5.34$, $p = 0.02$), VSi ($F(1,63)=7.30$, $p = 0.01$), VSs ($F(1,114)=9.12$, $p = 0.003$), mPFC ($F(1,56)=12.50$, $p < 0.001$), and dmPFC ($F(1,73)=4.27$, $p = 0.04$), and for all of these regions, the neural response was greater to Engaged replies than to Non-engaged replies. Several regions also showed a main effect of Partner Type. These regions included the amygdala ($F(1,90) = 4.19$ $p = 0.04$), VSi ($F(1,38)=7.59$, $p = 0.01$), VSs ($F(1,55)=7.52$, $p = 0.01$), RaSTS ($F(1,39)=15.73$, $p < 0.001$), LaSTS ($F(1,62)=9.73$, $p = 0.002$), mPFC ($F(1,38)= 15.32$, $p < 0.001$), and dmPFC ($F(1,105)=23.07$, $p < 0.001$), and for all of theses regions, the neural response was greater when receiving Peer replies than when receiving Computer replies. A subset of these regions, including amygdala ($F(1,114)=10.56$, $p = 0.002$), VSs ($F(1,114)=6.79$, $p = 0.01$), and dmPFC ($F(1,114) = 4.15$, $p = 0.04$), also showed an Engagement by Partner Type interaction, wherein the neural response to Peer Engagement was significantly greater than the neural response to both Computer Engagement and Peer Non-Engagement. The one region of interest for which there was a main effect of group (ASD vs. NT) during the reply period was the amygdala ($F(1,38)=4.54$, $p = 0.04$), which showed greater activation in NT participants compared to ASD participants. Figure 6 depicts amygdala activation during the reply period, where there was a significant main effect of group. See Appendices for graphs of the remaining ROIs.

Figure 6. Effect of mutual engagement: ROI analysis. During the reply period, the amygdala showed a significant main effect of group, with NT participants exhibiting greater activation than those with ASD across the Engaged and Non-Engaged conditions.



Exploratory Analysis: Relation Between Self-Reported Desire to See Peer Reply and Amygdala Activation. Given the amygdala's role in social reward and social cognitive processing, we were interested to see if amygdala activation during the initiation or reply period was related to participants' self-reported level of enjoyment when chatting with the peer. While NT participants' amygdala activation was not related to their self-reported enjoyment, participants with ASD showed a significant positive correlation between amygdala activation when receiving a reply from the peer (versus computer) and the degree to which they reported wanting to see the peer's (versus computer's) reply while chatting ($r(18) = 0.53$, $p = 0.02$) (Figure 7).

Figure 7. Participants with ASD showed a significant correlation between amygdala activation to Peer Reply > Computer Reply and self-reported desire to see Peer (versus Computer) replies.



Discussion

This study investigated the neural correlates of initiating and responding to bids for social interaction in children with and without ASD. Consistent with past work (Warnell et al., 2017), neurotypical participants exhibited greater activation in brain regions involved in social cognition and reward when they received responses from a peer than when they received responses from a computer. Surprisingly, participants with ASD overall had similar neural responses to their NT peers. They too exhibited greater activation to Peer versus Computer replies in several areas of the social brain as well as brain regions involved in reward processing. While the ASD group qualitatively exhibited less activation to Peer versus Computer replies compared to neurotypical controls, these differences did not reach significance at the whole brain level.

There are several possible reasons why we did not find significant group differences at the whole-brain level, as expected. First, ASD is a highly heterogeneous disorder, with highly heterogeneous neural activation patterns (e.g., Hasson et al., 2009). This variability may have minimized group differences in this relatively small sample. As noted in Figures 4 and 5, while group differences did not reach significance at the whole-brain level, NT and ASD participants displayed both overlapping and distinct activation when receiving replies from the Peer, with NT participants displaying greater activation in regions associated with reward processing (Yarkoni, Poldrack, Nichols, Van Essen, & Wager, 2011; www.neurosynth.org) such as mPFC, caudate, posterior cingulate, and amygdala. While Figures 4 and 5 do not represent statistically significant differences between groups, our statistical

thresholding and corrections for multiple comparisons at the whole-brain level (used to diminish Type I errors), may have increased the likelihood of Type II errors (Lieberman & Cunningham, 2009). Thus, Figures 4 and 5 may represent true differences between groups, but this will need to be replicated by future studies. As Lieberman and Cunningham (2009) note, it is better for neuroimaging researchers to report findings from more lenient statistical thresholds in order to avoid Type II errors (epecially because the cognitive, social, and affective neuroscience fields are still in an 'exploration phase'), and then rely on replication and meta-analysis to erase Type I errors. Otherwise, if we employ very stringent statistical thresholds to avoid Type I errors, we might miss true, subtle effects that will go unreported in meta-analyses.

It is possible that the current study may not have had enough power to detect group differences at the whole-brain level. While the number of participants in this study (i.e., 20 in each group) was larger than that of past studies investigating social motivation in ASD (i.e., an average of 16 in each group), it is possible that we still did not had enough power to detect differences between groups. This is especially true for interaction effects (Brookes et al., 2004), which were of primary interest in the current study. According to simulations reported in Brookes et al., (2004), a trial with 80% power for an overall effect only had 29% power to detect an equal magnitude interaction effect. To increase power for interaction effects, Brookes et al. (2004) suggest inflating sample sizes fourfold. Thus, future studies investigating neural circuitry in ASD using similar paradigms should significantly increase the sample size to ensure adequate power.

Another possibility is that the current findings represent true similarities between diagnostic groups. Despite the social cognitive weaknesses that are characteristic of ASD, many autistic individuals report wanting friends (Henault & Attwood, 2002); thus, they may have broadly intact neural reward circuitry when initiating and receiving replies from peers. This latter possibility has implications for how we understand the social motivation hypothesis of ASD.

Group differences did emerge, however, within the amygdala. We chose to include the amygdala as a region of interest due to its role in social cognition and social motivation (Berridge & Kringelbach, 2008; Chevallier et al., 2012; Dichter et al., 2012; Fareri, Martin, & Delgado, 2008; Haber & Knutson, 2010; Kohls et al., 2012; Schultz, 2005). During both the initiation and reply periods, participants with ASD showed a blunted amygdala response across conditions. Thus, when they were sharing information about themselves (with both the peer and computer) and when they were receiving information (from both the peer and the computer), participants with ASD showed *less* activation than their neurotypical counterparts.

The amygdala has been implicated in social cognition (Adolphs, 2010; Kennedy, Glascher, Tyszka, & Adolphs 2009; Adolphs & Spezio, 2006; Spezio, Huang, Castelli, & Adolphs 2007), and several studies have demonstrated differences in the amygdala between individuals with ASD and their neurotypical peers. Specifically, people with ASD show less amygdala activation when reading others' emotions (Baron-Cohen et al., 1999), show either increased (Dalton et al. 2005; Monk et al. 2010; Weng et al. 2011) or decreased (Critchley et al. 2000; Dapretto et al. 2006; Grelotti et al. 2005; Hadjikhani, Joseph, Snyder, & Tager-Flusberg, 2007)

amygdala activation to faces, have an altered growth pattern of neurons in the amygdala (Avino et al., 2018) that is related to symptom severity (Mosconi et al., 2009; Schumann, Barnes, Lord , & Courchesne, 2009), and have impaired connectivity between the amygdala and other key social brain regions (von dem Hagen, Stoyanova, Baron-Cohen, & Calder, 2013). The amygdala is also related to reward processing (Adolphs & Spezio, 2006; Klein et al., 2009) and shows abnormalities in individuals with ASD when processing social rewards (Dichter et al., 2012; Kohls et al., 2012).

Given the amygdala's role in social reward processing, we would expect greater activation in this region when participants were sharing self-relevant information with a Peer versus the Computer. However, neither NT nor ASD participants showed a differential neural response in this region when initiating a chat with the Peer versus the Computer. Instead, the NT group showed significant amygdala activation when sharing information with *both* Peer and Computer, while the ASD group did not show significant amygdala activation when initiating chats with *either* conversational partner. There are two plausible reasons for this finding. First, the nature of the task may have been too cognitively overwhelming for participants to distinguish between Peer and Computer conditions during the initiation period. Specifically, they were asked to quickly respond to a prompt (e.g. "I have been to Baltimore") and may not have had time to notice whether they were chatting with the Peer or with the Computer (which was noted at the top of their screen). We believe that this is the most likely explanation. A second possibility is that participants actually found the two conditions equally rewarding. We believe this is

unlikely, given that both NT and ASD participants reported that they enjoyed sharing information about themselves with peers significantly more than they enjoyed sharing information with the computer.

Thus, greater amygdala activation across both conditions (Peer and Computer) in NT participants during the initiation period likely reflects a neural correlate of initiating social interactions by sharing self-relevant information, which is a critical component of normative social development (Buhrmester & Prager, 1995; Collins & Miller, 1994; Sprecher, Treger, Wondra, Hilaire, & Wallpe, 2013). Children and adolescents with ASD, on the other hand, have characteristic weaknesses in social initiation (Kamps et al., 1992; Koegel, Koegel, Frea, & Fredeen, 2001; Oke & Schreibman, 1990; Shabani et al., 2002; Strain, Kerr, & Ragland, 1979), which may have been reflected in the current study by underactive amygdala responses when sharing information about themselves.

Group differences in the amygdala also emerged during the Reply period of the task, when participants received information from the peer (i.e., "Me too!" or "I'm Away") and computer (i.e., "Matched! or "Disconnected"). At the whole-brain level, NT participants showed significant activation in bilateral amygdala when receiving an engaged response from a peer (e.g., "Me too!") versus receiving a non-engaged response from a peer (e.g., "I'm Away"). Participants with ASD did not show activation in amygdala to this contrast, although between-group differences did not reach significance at the whole-brain level. Amygdala ROI analyses revealed a significant main effect of group, with NT participants exhibiting greater activation than participants with ASD regardless of whom they were receiving a response from

(Peer or Computer) and regardless of whether or not the conversational partner was engaged ("Me too!" or "Matched!") or not engaged ("I'm Away" or "Disconnected"). Further supporting the role of the amygdala in social reward processing, participants with ASD demonstrated a significant correlation between amygdala activation to Peer (versus Computer) replies and self-reported desire to see the Peer's (versus Computer's) reply. NT participants did not show this correlation, possibly due to limited variability in their responses to the post-scan questionnaire.

The only other group difference that emerged was in the right TPJ during the initiation period. Unexpectedly, participants with ASD showed significantly *higher* activation than NT participants in this region while sharing self-relevant information with the peer and the computer. The TPJ is involved in thinking about other people (i.e., mentalizing; Decety & Lamm, 2007; Samson, Apperly, Chiavarino, & Humphreys, 2004; Saxe & Kanwisher, 2003) as well as thinking about oneself (Vogeley & Fink, 2003). A large body of literature shows reduced activation in TPJ in ASD during tasks that involve mentalizing, or taking the perspective of another person (Castelli et al., 2002; Kana et al., 2009; Lombardo et al., 2011; Murdaugh, Nadendla, & Kana, 2014; but see Dufour et al., 2013). One study investigating theory of mind found TPJ *hyper*activation in ASD (Mason, Williams, Kana, Minshew, & Just, 2008). Greater activation in a brain region associated with a core area of dysfunction in ASD, such as theory of mind, may reflect a compensatory mechanism (see Dichter, Felder, & Bodfish, 2009; Schmitz et al., 2006). In fact, research in disorders such as schizophrenia and depression suggests that psychopathology can be associated with hyperactivation of brain regions that are related to the disorder

(Buchsbaum et al., 2007; Manoach, 2003; Wagner et al., 2006), supporting the notion of cortical inefficiency. In the current study, it is therefore plausible that participants with ASD required more TPJ activation than their neurotypical peers when initiating conversations, because this is a core area of dysfunction in the disorder (Kamps et al., 1992; Koegel et al., 2001; Oke & Schreibman, 1990; Shabani et al., 2002; Strain, Kerr, & Raglan, 1979).

Overall, findings suggest that, despite the centrality of social interactive difficulties in ASD, neural differences between children and adolescents with ASD and their neurotypical peers are subtle. There were no significant whole-brain differences between groups during the Initiation or Reply periods, and both groups recruited social cognitive and reward circuitry while interacting with a peer. However, the whole-brain response to peer replies qualitatively appeared more robust in the neurotypical sample, and ROI analyses supported significant between-group differences in the amygdala and right TPJ.

Conclusion

In sum, this study was the first to compare the neural correlates of participants with and without ASD during a naturalistic "chat" paradigm. Findings are broadly consistent with Dufour et al. (2013), who found minimal differences during theory of mind tasks in a very large sample of adults with and without ASD. It is possible that group differences in the current study could be obscured due to the highly heterogeneous nature of the ASD sample; for example, different individuals may have different neural correlates of social interactive challenges. It is also possible that social interactive difficulties could be present in individuals with ASD without

corresponding differences in brain activation. Future studies should examine other neural indices of social interaction such as connectivity between regions and should examine the contribution of factors such as age.

Appendices

Post-Scan Questionnaire

CHT_ASD Rating Questionnaire ID: Date:

CHT Parameters: _____

Pre-test (after they select their chat partner during mock)—REMEMBER TO SHOW SCALES TO CHILD

1) How much are you interested in chatting with _____? Where 1 is not at all and 5 is a lot.

   _____

2) How much do you think you'd like _____ in real life? Where 1 is not at all and 5 is a lot?

   _____

Post-test—PULL A PHOTO UP OF THE CHAT PARTNER BEFORE THE CHILD COMES IN TO THE ROOM. USE THE CORRECT SCALES.

First, let's talk about _____. (Fill in name)

1) How much did you like chatting with _____? Where 1 is not at all and 5 is a lot.

   _____

2) How much do you think you'd like _____in real life? Where 1 is not at all and 5 is a lot?

   _____

3) Sometimes it can be hard to pay attention when you're playing games. How much did you pay really close attention answering a question when he/she was the one you were chatting with? Where 1 is not at all and 5 is a lot.

   _____

4) How much did you want to see his/her answer to your question? Where 1 is not at all and 5 is a lot.

   _____

5) How did you feel when she/he agreed with your answer? Where 1 is very bad and 5 is very good.

_____

6) How did you feel when she/he disagreed with your answer? Where 1 is very bad and 5 is very good.

_____

7) How did you feel when she/he was away and didn't respond? Where 1 is very bad and 5 is very good.

_____

8) What did you like the most?

   ANSWERED AWAY THE SAME

9) How much do you think _____ would want to be your real-life friend? Where 1 is not at all and 5 is a lot.

_____

10) Tell me about what kind of person you think _____is.

_____
_____
_____

11) Why do you think _____was away for some questions?

_____
_____
_____

Now let's talk about those questions where no one saw your answer and it was just the computer.

12) How much did you like it when you were just answering the computer? Where 1 is not at all and 5 is a lot?

_____

13) Sometimes it can be hard to pay attention when you're playing games. How much did you pay really, really close attention answering a question when you were chatting with the computer? Where 1 is not at all and 5 is a lot.

_____

14) How much did you want to see if the computer matched your answer? Where 1 is not at all and 5 is a lot.

_____

15) How did you feel when your answer matched the random answer? Where 1 is very bad and 5 is very good.

_____

16) How did you feel when your answer did not match the random answer? Where 1 is very bad and 5 is very good.

_____

17) How did you feel when the computer was disconnected? Where 1 is very bad and 5 is very good.

_____

18) What did you like the most?

CONNECTED (told you matched or mismatched) DISCONNECTED

THE SAME

19) Do you think there was more to this game than we told you about?

YES NO

20) [If yes] What?

_____
_____
_____

21) Did you like chatting with a person or the computer more?

PERSON COMPUTER

22) Did you pay more attention when it was a person or the computer?

PERSON COMPUTER

23) When you were connected to the computer, did anyone see your answer?

YES NO

24) When the other person was playing the maze, did they still see your answer?

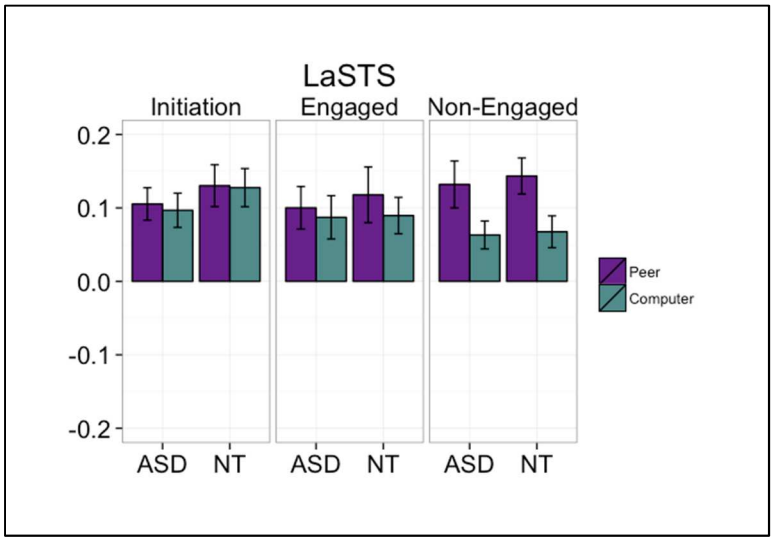
YES NO

25) Were there any particular questions that were fun to answer or hard to answer?

_____
_____
_____

26) Is there anything else you want to tell us about the chat task?

Supplemental Graphs: ROIs


LaSTS
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Engaged
Non-Engaged
0.2
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-0.1
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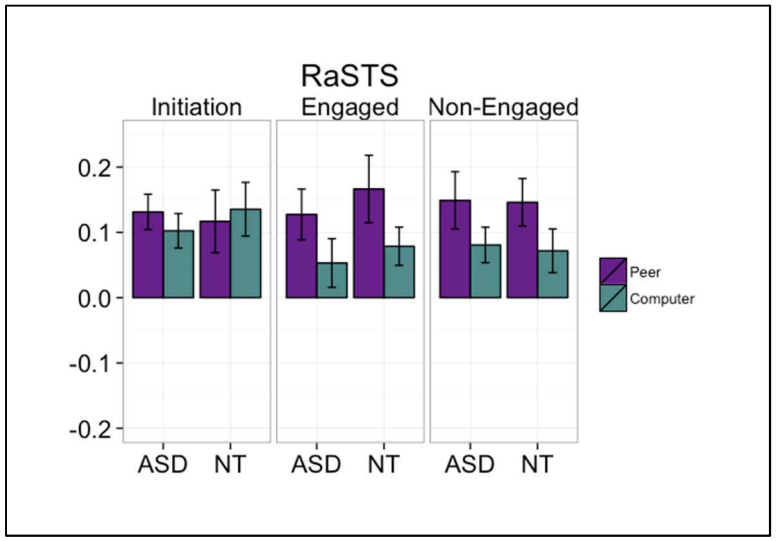
ASD
NT
Peer
Computer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Peer
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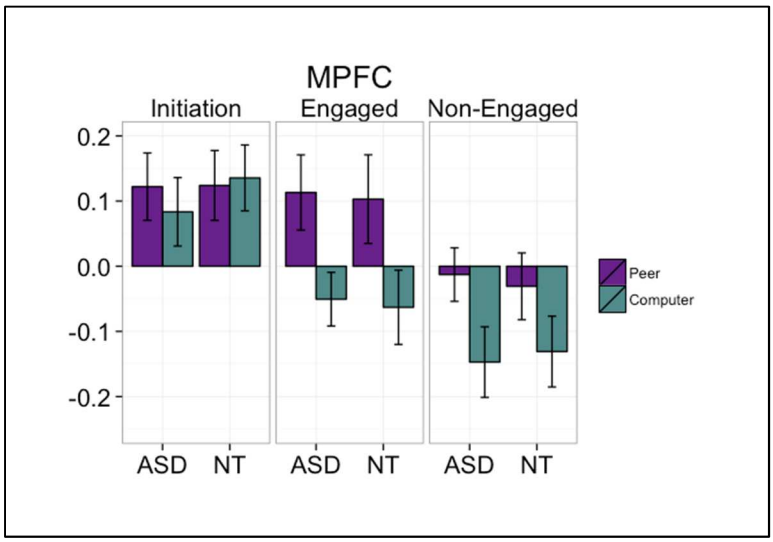
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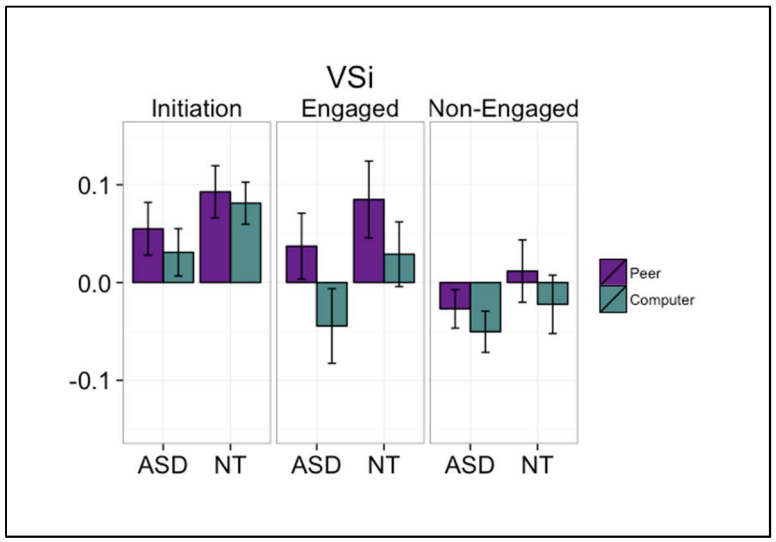
ASD
NT
Peer
Computer


VSi
Initiation
Engaged
Non-Engaged
0.1
0.0
-0.1
ASD
NT
Peer
Computer

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